

Letter to the Editor

Fetal Haemoglobin level in Pre-eclampsia

The Editor

Sir,

Pre-eclampsia still remains an enigmatic, multi-system and multi-factorial disease entity. So many hypotheses have been put forward to explain the origin and pathophysiology of this disease that it has been termed a “disease of theories” (1). One of these theories is the increase in the transfer of fetal red blood cells into maternal circulation due to impaired uteroplacental circulation and severe fetal growth restriction (2–4).

The authors conducted a prospective, case-control study in which 25 consecutive, pre-eclamptic patients admitted to the antenatal ward of the University Hospital of the West Indies, Kingston, were studied. An equal number of non pre-eclamptic, gestation-matched women attending the antenatal clinic of the same hospital served as controls. The subjects and controls were all nulliparous, between 28 and 40 weeks of gestation, and 18 and 39 years of age.

The diagnosis of pre-eclampsia was made when the patient had a blood pressure reading above 140/90 mmHg or an increase of 30 mmHg systolic and 15 mmHg diastolic over the booking blood pressure and with proteinuria on dipstick of one plus (+) or more. Patients with diabetes mellitus, liver or kidney disease were excluded. The study was explained to the subjects and their written consent obtained before recruitment into the study which was approved by the Ethical Committee of the University of the West Indies Faculty of Medical Sciences/University Hospital of the West Indies, Kingston, Jamaica.

Fetal haemoglobin level was estimated by the alkaline denaturation method (modified Betke method) as described by Dacie and Lewis (5). Briefly, sodium hydroxide was added to the lysate and after two minutes, the denaturation of haemoglobin was stopped by adding saturated ammonium sulphate. This precipitated the denatured haemoglobin. After filtration, the quantity of undenatured (unprecipitated) haemoglobin was measured. The proportion of alkali-resistant (fetal) haemoglobin was then calculated as a percentage of the total amount of haemoglobin present. There was no statistically significant difference in the HbF levels between

the pre-eclamptic ($1.73 \pm 0.16\%$) and control ($1.72 \pm 0.20\%$) groups.

Contrary to the afore-mentioned reports (2–4), we did not observe any significant increase in the level of fetal haemoglobin in the pre-eclamptic women compared with the controls. One possible explanation for this discrepancy might be the severity of the disease. The pre-eclamptic women in this study had only moderate pre-eclampsia, while those studied by the previous authors had more severe pre-eclampsia. Another reason could be the presence of fetal growth restriction. In fetal growth restriction, there is increased production of erythropoietin and erythroblastosis. The proportion of fetal erythroblasts was 5.5% in the pre-eclamptic patients who subsequently developed fetal growth restriction (3) and 8.5% in pre-eclamptic women with severe fetal growth restriction (4). It is therefore likely that the severity of the disease and the presence of fetal growth restriction might have resulted in a greater impairment of the implantation process and hence an increase in the transfer of fetal red blood cells into the maternal circulation.

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